

ANIMAL HEALTH SURVEILLANCE

Information contributed by staff of the Livestock Health and Pest Authorities and the NSW Department of Primary Industries

Clinical bluetongue exclusion: photosensitisation

A hobby farmer near Ballina reported to veterinary authorities that two of his 14 sheep had weakness, lameness, drooling, and sores on the mouth and nose.

The sheep were grazing naturalised, lush subtropical pasture, including *Setaria* sp., and were occasionally fed wheat.

The district veterinarian found that both sheep were febrile and had swollen ears, conjunctivitis and crusting on the nose. One sheep had crusting and sores around the perineum. The other had an inflamed tongue tip and a 1.5 by 0.4 cm rectangular-shaped lesion on the surface of the dental pad. The lesion was covered in greyish yellow closely adherent necrotic tissue, which when removed revealed a red, fresh, shallow wound.

The State Veterinary Diagnostic Laboratory confirmed that the lesions were consistent with liver damage and secondary photosensitisation, possibly caused by the fungus *Pithomyces*, which could have accumulated to high levels in the pasture because of recent humid weather. The Australian Animal Health Laboratory in Geelong excluded both bluetongue and foot-and-mouth disease. One sheep had been exposed to bluetongue virus in the past. The other was antibody negative for the virus, and both were PCR (polymerase chain reaction) negative.

The subtropical North Coast of NSW is not a major sheep producing

region: it has about 5000 sheep on 350 properties, where they are kept as pets and to manage pasture growth on small holdings or plantations. In comparison, there are more than 370,000 beef cattle in the region.

It is unusual to detect bluetongue antibody in North Coast sheep. *Culicoides brevitarsis*, the midge vector for bluetongue virus in this region, preferentially feeds on cattle. A survey done on the North Coast in the late 1970s found no sheep to be antibody positive. Clinical investigations, such as the one outlined in this case study, provide evidence that Australia's sheep population is free from clinical bluetongue, despite the circulation of various serotypes in cattle. The National Arbovirus Monitoring Program (NAMP) provides detailed information about the distribution of bluetongue virus in Australia.

**For further information contact
Matt Ball, Senior District Veterinarian,
North Coast Livestock Health and Pest
Authority, Lismore, on (02) 6621 2317.**

Foot-and-mouth disease excluded – salmonellosis suspected

Two cows in a mob of 35 AIS (Australian Illawarra Shorthorn) cows and calves died on the North Coast after showing reluctance to move, drooling of saliva, mild scour and inappetence. Stillbirth

In this issue!

Clinical bluetongue exclusion: photosensitisation	1
Foot-and-mouth disease excluded – salmonellosis suspected	1
Iodine deficiency in lambs	2
Whatever happened to mad cow disease?	3
Endocarditis and liver damage in a cow	4
TSE excluded in death of sheep	5
Leptospirosis in a beef herd	5
Osteoporosis in lambs	5
Seasonal swayback in sheep near Lake Cargelligo	5
Ill-thrift syndrome in a well-managed beef herd	6
Suspected lead poisoning	6
Benign theileriosis in beef calves	6
Calf diphtheria in Hereford calves	7
Lead poisoning in yearling cattle	7



Jaundice and haemorrhages in a suspected case of salmonella septicaemia. Photo: M. Ball

was observed in two other cows, and two newborn calves were also found dead.

The district veterinarian examined a live, sick animal, which had fever, dehydration, scant smelly and mucous scour and a mild purulent discharge from the vulva. The mouth was normal. The cow was not lame and had no feet lesions. The urine was dark but not red. The cow died the next morning and post-mortem examination showed an enlarged liver and jaundice. The jejunum and ileum were gas filled and in some areas contained haemorrhage. The epicardium had significant ecchymotic haemorrhages. Similar haemorrhages were seen on the pleural surface of a few lung areas. The spleen, although not grossly swollen, had a jam-like consistency on the cut surface.

Laboratory examination of a range of tissue samples suggested an inflammatory response and liver damage, but no bacteria were isolated and tick fever was excluded. Samples sent to AAHL (Australian Animal Health Laboratory) were all negative for foot-and-mouth disease tests.

Although laboratory culture of bacteria was negative, the district veterinarian diagnosed salmonellosis on clinical and epidemiological evidence. Different mobs had recently been mixed together and placed in a paddock that had been heavily contaminated with faeces. Spread of salmonella from a stressed carrier or from

point-source environmental contact was likely.

The farmer was advised to increase stock observation, especially around the time of calving, and to seek private veterinary treatment of any sick stock.

**For further information contact
Matt Ball, North Coast Livestock
Health and Pest Authority, Lismore, on
(02) 6621 2317.**

Iodine deficiency in lambs

Iodine deficiency was diagnosed as the cause of goitre (enlarged thyroid gland) and mortalities in neonatal lambs in a small flock of Dorper ewes and lambs near Condobolin. Within 1 week, six lambs were either born dead or were very weak at birth and then died within the first few days of life.

A dead lamb and a live lamb were examined by the district veterinarian. The live lamb had a sparse hair/wool cover over its body, hairless ears, and an obvious swelling around the throat area. The dead lamb had a similar hair pattern and a 6-cm-long swelling in the neck. A post mortem revealed this to be an enlarged thyroid gland. An ochre-coloured liver was also noted.

The laboratory diagnosed hyperplastic goitre and a suppurative, multifocal, minimal, subacute hepatitis.

Diagnosis of goitre is usually made on the basis of an assessment of the thyroid weight in comparison with body weight, with a thyroid weight greater than 0.3% of body weight being definitive. The size of the thyroid gland in the lamb more than likely exceeded this threshold.

Goitre is generally the result of iodine deficiency or the consumption of goitrogenic plants, such as brassica fodder crops that block iodine uptake. Iodine deficiency is most commonly seen on sandy soils and when there is lush pasture growth, as the amount of surface soil consumed is reduced. Although goitre is occasionally seen in goats in the Condobolin area, it is rarely seen in lambs. The ewes did not have a history of grazing goitrogenic plants. However, the ewes were grazing on sandy river flats, so it is probable that these soils were iodine deficient.

No further cases occurred after lick blocks with a high iodine content were provided.

**For further information contact
Katharine Marsh, District Veterinarian,
Lachlan Livestock Health and Pest
Authority, Condobolin, on (02)
6895 2152.**

Whatever happened to mad cow disease?

Reg Butler from the Commonwealth Department of Agriculture, Fisheries and Forestry (DAFF) has provided a summary of information on what used to be called 'mad cow disease'. Surveillance for this disease still occurs in Australia.

Classical bovine spongiform encephalopathy (C-BSE), the proper name for mad cow disease, is a non-contagious transmissible spongiform encephalopathy (TSE) that primarily affects cattle. It originated in the UK in the mid-1980s and spread to several countries through infected live cattle and contaminated meat and bone meal. Cases of C-BSE were found in indigenous cattle in mainland Europe, Japan, Canada and Israel.

Primary variant Creutzfeldt-Jakob disease (vCJD) in people is caused by eating hazardous tissues (e.g. central nervous system [CNS] tissue) from C-BSE-infected cattle. The total number of definite or probable vCJD cases recorded in the UK is 176.

The vCJD outbreak in the UK is now in decline, albeit with a lengthy tailing-off

period. The peak number of deaths was 28 in 2000, and there were 3 cases in 2010.

Domestic cats, some zoo animals, two goats and non-human primates have also acquired TSE through eating C-BSE-contaminated food.

BSE has never been recorded in Australia. Scrapie has occurred once, in imported sheep on a single property in 1952. It was promptly eradicated.

Three strains of BSE have been identified to date. Widespread global surveillance for C-BSE has identified two additional and very rare strains, called low-type (L-type) BSE and high-type (H-type) BSE because the abnormal prion proteins are different from that in C-BSE. L-type BSE and H-type BSE are also collectively referred to as 'atypical BSE' and appear to be epidemiologically unrelated to C-BSE. One hypothesis is that they are sporadic or genetic diseases in aged cattle.

Challenge studies overseas have shown that both atypical strains can infect cattle. Further studies are under way to determine whether tissues other than the CNS in infected cattle are potential sources of infection and whether these atypical strains can be orally transmitted to cause disease in livestock or humans.

Unlike atypical BSE, atypical scrapie is not infective and poses no threat to humans. Atypical scrapie has been found in most scrapie-free countries that test large numbers of sheep for scrapie. It is thought to be a rare, sporadic degenerative disease of the brain that occurs spontaneously in older sheep and less commonly in goats. To date, two cases of atypical scrapie have been found in Australia. This disease is thought to be spontaneous and sporadic, and these two cases had no relationship to each other.

The OIE (World Organisation for Animal Health) sets recommended risk-reduction measures for BSE. Since the use of animal by-products as stock feed components was banned, the numbers of cases of BSE have declined dramatically around the world. The BSE risk status of the Australian cattle



Enlarged thyroid glands in a neonatal lamb. Photo: K. Marsh

herd has been protected through a ban on imports of meat and bone meal (except from New Zealand) since the 1960s, and more recently by controls on imports of live cattle, stockfeed, stockfeed ingredients and veterinary therapeutics.

On the latter point, veterinary vaccines and other biological products are considered to be high risk. For this reason very stringent precautions are taken to ensure that all components of such biological products are TSE free. DAFF has recently released *Draft guidelines for managing the risk of transmitting TSEs via veterinary vaccines and other in vivo veterinary products* for consultation. This document can be accessed at <http://www.daff.gov.au/ba/ira/current-animal/tses>

NSW continues to implement the NTSESP (National TSE Surveillance Program), which implements the sampling and testing of cattle and sheep with TSE like signs. Five months into the financial year, we had 57 sheep and 55 cattle submissions. Our end-of-year target is 149 sheep and 87 cattle. Figures to date show that Livestock Health and Pest Authority district veterinarians account for the majority (70%) of sheep submissions, whereas the majority (64%) of cattle submissions come from private practices.

NTSESP provides owner incentives of \$300 and \$50, respectively, for cattle and sheep samples fitting the surveillance criteria. Veterinarians receive \$200 and \$100, respectively, for cattle and sheep

samples, in addition to a collection/documentation fee of \$100. The additional bonus for both owner and veterinarian is that the laboratory cost of identifying the cause of death or sickness is free of charge. Veterinarians are therefore encouraged to use the TSE program, wherever possible, on eligible animals.

For further information, contact Dermot McNerney, State Residues Coordinator NSW DPI, Dareton, on (03) 5019 8411.

Endocarditis and liver damage in a cow

Recumbency, jaundice and chronic mastitis were seen in a cow that had arrived from a known theileria-infected herd. On post-mortem examination the cow had ascites as well as a classic 'nutmeg' liver due to chronic passive congestion, which accentuates the lobular pattern, with red central and yellow or tan periportal zones. There was also a massive vegetative endocarditis on the valves in the right ventricle. It is likely that the bacteria causing the mastitis also caused the valvular endocarditis, which in turn caused congestion in the liver and hepatic anoxia.

For further information contact Bob McKinnon, Senior District Veterinarian, Central North Livestock Health and Pest Authority, Tamworth, on (02) 6762 2900.



Chronic passive venous congestion of the liver. Photo: B. McKinnon

TSE excluded in death of sheep

TSE was excluded as the cause of neurological disease and death in sheep near Young. In late August 2011 adult ewes were found with an abnormal gait, and a few died. They were removed from the paddock, but clinical disease and losses continued. The ewes staggered, particularly when moved, and subsequently became recumbent, with opisthotonos and paddling when disturbed.

A post-mortem examination performed on two affected ewes in November 2011 revealed a green line in the kidneys of both sheep, consistent with phalaris grass toxicity. Histopathology confirmed that the brainstem was grossly pigmented with abundant intraneuronal pigment, consistent with ingestion of phalaris. Histopathology also ruled out TSE as the cause of disease.

The ewes had been on phalaris-dominant pasture in the period before they showed clinical disease. Despite being moved off this pasture, the flock continued to develop clinical disease for a further 2 months.

For further information contact Elizabeth Braddon, Senior District Veterinarian, Lachlan Livestock Health and Pest Authority, Young, on (02) 6382 1255.

Leptospirosis in a beef herd

Leptospirosis was determined to be the cause of 30% of calf losses in a beef herd at Lake Cargelligo in October 2011. In a herd of 80 cows pregnancy-tested as in calf in the post-joining period, 28 (35%) were dry at calf marking. Two dead calves were seen during the calving period, but all other calves appeared normal on paddock inspections.

Blood and hair samples were collected at a property visit from both the dry and the wet cows. All cattle were in good physical condition and health. The blood results showed that the dry cows had *Leptospira pomona* titres well above normal, with most titres being 3200 or greater (range 800 to

>3200), whereas titres in the wet cows with calves were essentially within normal limits. Pestivirus test results indicated past exposure but no current activity.

This herd was not routinely vaccinated pre-calving for clostridial and leptospiral infections. The rainy season supported high mice and feral pig populations and was wet enough to cause water pooling in low areas. The cause of the outbreak in this case was exposure of a naïve herd to a particularly high environmental challenge of *L. pomona*.

The owner was advised to vaccinate the cattle herd with 7-in-1 vaccine pre-calving to prevent a future outbreak.

For further information contact Elizabeth Braddon, Senior District Veterinarian, Lachlan Livestock Health and Pest Authority, Young, on (02) 6382 1255.

Osteoporosis in lambs

Ten out of 400 weaner lambs were found in recumbency due to fractures of the long bones. The fractures had occurred while the lambs had been walking from the paddock into the yards. On close inspection up to 10% of the flock had some form of lameness. The weaners had been grazing an oat paddock and had been given a lupin supplement.

On post-mortem examination the weaners had fractures of the long bones, along with multiple fractures along the ribs. The ribs were rubbery and the vertebrae could be halved with slight pressure from a knife.



Bone fracture and blood clot due to thin cortical bone in a lamb. Photo: B. McKinnon

Test results indicated that the animals were hypocalcaemic, and one of the weaners was also copper deficient.

The owner treated the rest of the flock with a mineral supplement, used a calcium dry lick to balance the calcium level in the oat crop and the lupins, and fed some hay to ensure adequate vitamin D intake.

For further information contact Bob McKinnon, Senior District Veterinarian, Central North Livestock Health and Pest Authority, Tamworth, on (02) 6762 2900.

Seasonal swayback in sheep near Lake Cargelligo

In late spring, swayback was responsible for ataxia and recumbency in a 6-week-old lamb in a mob of approximately 500 ewes and lambs grazing saltbush, barley grass, clover and crowfoot in the Lake Cargelligo region. The lamb walked with a distinct side-to-side swaying motion for a few seconds before its legs knuckled and it fell to the ground.

Serum copper levels were very low at 1.7 mmol/L (normal 7.5 to 20 mmol/L). In the spinal cord there was myelin degradation and an influx of inflammatory cells. A few swollen axons were also present randomly in the white matter.

There are two main causes of copper deficiency in stock: primary deficiency from low concentrations of copper in the pasture and secondary copper deficiency caused by ingestion of excessive levels of molybdenum and sulfur in plants or soils. These bind copper in a highly insoluble compound that is not absorbed in the intestine.

Copper deficiency is more prevalent during periods of lush growth of green feed. A marginal deficiency detected in mid-spring is usually resolved by mid-summer as the pasture dries off. In this case, the high demand for copper coincided with lush feed and relatively low copper status.

Treatment in lambs affected in this way is not recommended. No further action was taken by the owner, as no further clinical cases were noticed.

For further information, contact Dermot McNerney, State Residues Coordinator NSW DPI, Dareton, on (03) 5019 8411.

Ill-thrift syndrome in a well-managed beef herd

A herd of Hereford-cross mature cows and yearlings on lush, fertilised pastures on the North Coast suffered acute diarrhoea with significant weight loss over a 2-week period. They had received no recent mineral supplementation in an area noted for moderate copper and selenium deficiency. They had not been drenched for at least 3 or 4 months before the onset of clinical signs.

On clinical examination, some of the yearlings had a 'washed-out' coat colour indicative of copper deficiency. Some cows were continuing to scour (but not severely), and some had low body-condition scores despite the good pasture.

A full range of specimens was taken from a sample of 7 yearlings and 5 cows for laboratory examination.

Laboratory and clinical findings supported a diagnosis of complex mineral deficiency combined with intestinal parasitism with *Ostertagia*.

Of particular note was an apparent iodine deficiency showing up as low thyroxine hormone levels. Iodine deficiency has not been regularly detected on the North Coast, and there has been no history of clinical goitre or alopecia syndromes to indicate significant problems. It is likely, however, that iodine levels are marginal or deficient more commonly than is recognised. If so, they may in fact be compounding recognised ill-thrift and reduced-performance syndromes that are more commonly attributed to copper or selenium deficiency.

Also, blood thyroxine levels could have been exacerbated during the 2011 winter-spring on the North Coast because of the particularly vigorous growth of white clover in the pasture; this may have made iodine unavailable for incorporation into the hormone.

For further information contact David Thomson, North Coast Livestock Health and Pest Authority, Grafton, on (02) 6642 3699.

Suspected lead poisoning

Sudden onset of grand mal seizures occurred in a 4-month-old Jersey heifer in a commercial dairy herd on the North Coast. It was unaware of its surroundings and apparently blind. It had access to an old cottage that had been damaged by a storm the year before, when the door had blown in and the roof had blown off. Old white paint was peeling from the ceiling under which the calf had been camping. The district veterinarian made a provisional diagnosis of lead poisoning and euthanased the calf.

Laboratory analysis of kidney samples confirmed marginally high lead concentrations.

The remaining calves were removed from the paddock to ensure that they had no further access to the chemically affected area. They were detained for testing in 12 months' time to ensure that there are no lead residues in offal from any stock slaughtered for human consumption.

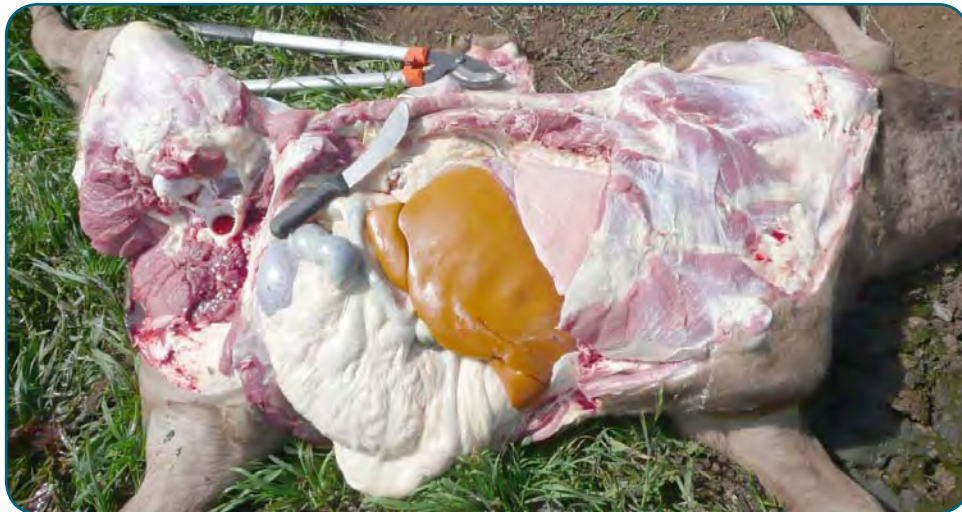
For further information contact Phil Kemsley, North Coast Livestock Health and Pest Authority, Casino, on (02) 6662 3166.

Benign theileriosis in beef calves

In October 2011, a 400-cow beef breeder herd on the eastern escarpment of the Northern Tablelands had five calves, aged 12 weeks, die within a week. They were all part of one of nine separately grazed mobs. One sick calf was found recumbent and depressed, with very pale mucous membranes and a temperature of 41.8 °C.

The district veterinarian conducted a post-mortem examination on a recently deceased calf. The carcass was very pale. The liver was approximately 1.5 times the normal size and was a diffuse bright ochre colour. There was no generalised jaundice of the carcass. The heart had splash haemorrhaging across its surface. The blood appeared not to clot and there was no heart clot present. The spleen was enlarged.

Blood smears revealed that *Theileria* sp. was present in less than 1% of red blood cells. The blood PCV (packed cell volume)



Pale carcass with distinct, swollen, ochre-coloured liver. Photo: S. Eastwood

was 8.0% (normal range 20% to 50%). Liver histology on samples from the dead calf indicated bilirubin accumulation. Marked hepatocyte injury was not noted, suggesting that haemolysis was the likely cause of the ochre colour in the liver.

Benign theileriosis was diagnosed, even though the percentage of *Theileria* organisms found in the red blood cells was classed as 'rare'. This is often the case in acutely ill animals and is exacerbated by the anaemia.

The mob was treated with an acaricide to minimise bush tick infestation and no further cases occurred.

For further information contact Steve Eastwood, Senior District Veterinarian, New England Livestock Health and Pest Authority, Armidale, on (02) 6772 2366.

Calf diphtheria in Hereford calves

Two calves approximately 2 months old in a mob of 25 Hereford cows with calves at foot in the Bathurst region were noticed to be sick. Both calves were febrile, dyspnoeic and snoring. Inspiration could be obstructed by gently pressing on the lateral walls of the larynx. Calf diphtheria was diagnosed by the district veterinarian on clinical grounds.

Penicillin therapy was effective in one calf, but the other died. At necropsy the internal mucosa of the larynx was lined with a

5-mm-thick layer of yellow, tightly adherent caseous material. The surrounding tissues were hyperaemic, and some ingesta were lodged anterior to the diphtheritic ring within the larynx.

Calf diphtheria is caused by *Fusobacterium necrophorum*, a widespread anaerobic bacterium inhabiting the gut in herbivores and excreted in the faeces. It typically affects the larynx and pharynx of calves that are raised under unsanitary conditions or that are subject to stress or concurrent disease.

For further information contact Bruce Watt, Senior District Veterinarian, Tablelands Livestock Health and Pest Authority, Bathurst, on (02) 6331 1377.

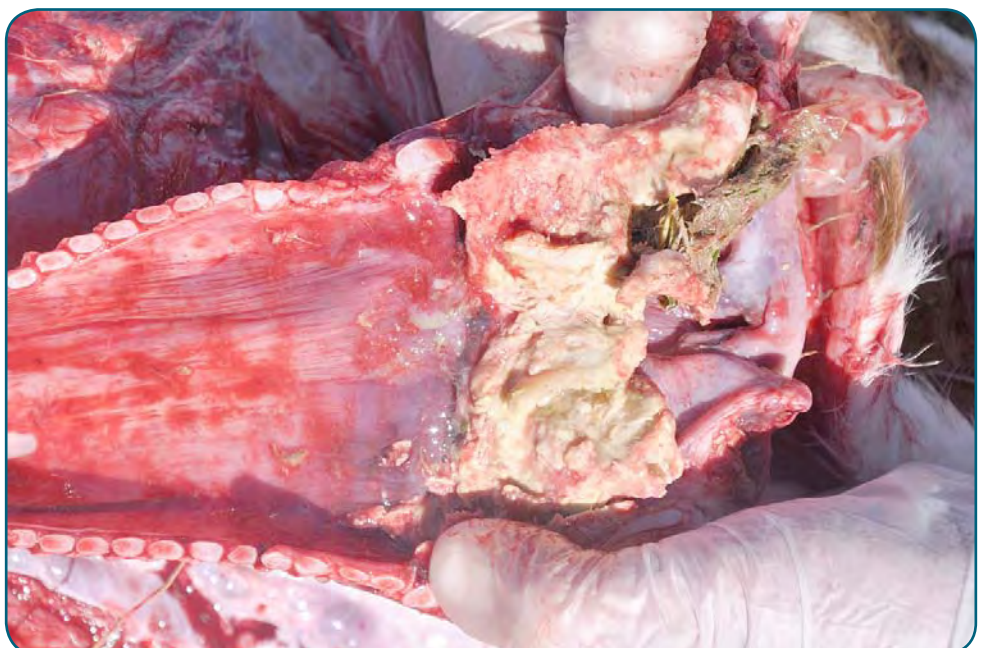
Lead poisoning in yearling cattle

Four heifers died from among a mixed mob of 45 yearlings grazing a sparse, mature oat crop on sandy soil near Pilliga, NSW. Before they died the calves showed bizarre behaviour, including chewing on wood and bags but not on the commercial lick that was on offer.

One heifer, which had died in sternal recumbency, had blood oozing from her nose. Her conjunctivae were swollen and congested with blood. There were no signs of struggle. Before necropsy, an anthrax ICT (immunochromatographic test) was conducted, with a negative result. During necropsy, a substance that looked similar to lead was found in the rumen; lead toxicity was later confirmed via laboratory analysis of kidney tissue. Subsequently, the owner found the source of the lead to be the remains of a car battery in a pile of burnt rubbish in the paddock.

Following confirmation of lead toxicity, the remaining yearlings were subject to regulatory restrictions because of the potential presence of lead residues.

For further information contact Libby Read, District Veterinarian, North West Livestock Health and Pest Authority, on (02) 6792 2533.



A diphtheritic membrane lined the larynx adjacent to the inflamed trachea in this calf. Photo: B. Watt

Getting information on animal diseases

This surveillance report can convey only a very limited amount of information about the occurrence and distribution of livestock diseases in New South Wales. If you would like more specific information about diseases occurring in your part of the State, contact your local Livestock Health and Pest Authorities District Veterinarian or Departmental Regional Veterinary Officer.

For Statewide information, contact the NSW Department of Primary Industries Animal and Plant Biosecurity Branch in Orange on (02) 6391 3237 or fax (02) 6361 9976.

For more information on national disease status, check the National Animal Health Information System (NAHIS) via the internet at:
<http://www.animalhealthaustralia.com.au/status/nahis.cfm>

This is a report under the Animal Disease Surveillance Operational Plan, Project 8, 'Reporting for Animal Disease Status in NSW'.

Prepared by Rory Arthur, Animal and Plant Biosecurity Branch,
NSW Department of Primary Industries, Kite St, Orange 2800.
Phone (02) 6391 3806

E-mail: rory.arthur@industry.nsw.gov.au

**Copies of NSW Animal Health Surveillance reports are available on the internet at:
<http://www.dpi.nsw.gov.au/newsletters/animal-health-surveillance>**

Disclaimer

The information contained in this publication is based on knowledge and understanding at the time of writing (January 2012). However, because of advances in knowledge, users are reminded of the need to ensure that information upon which they rely is up-to-date and to check the currency of the information with the appropriate officer of NSW Department of Primary Industries or the user's independent adviser.

The product trade names in this publication are supplied on the understanding that no preference between equivalent products is intended and that the inclusion of a product name does not imply endorsement by the NSW Department of Primary Industries over any equivalent product from another manufacturer.



Department of
Primary Industries



LIVESTOCK HEALTH AND PEST AUTHORITIES

LHPA
Safeguarding Agriculture in NSW